PHILADELPHIA NEUROLOGICAL SOCIETY.

February 26, 1900.

The President, Dr. William G. Spiller, in the chair.

TWO CASES OF TABES DORSALIS IN NEGROES—HUSBAND AND WIFE.

Dr. A. P. Francine presented a husband and wife, of negro blood, with tabes. The man was 48 years of age. He positively denied the presence of white blood in his family. He had had a chancre when 17 years old, and was married to his present wife in 1888. Five years ago vision began to fail. The diagnosis of tabes was made on account of ataxia, shooting pains, numbness in the soles of the feet, girdle sensation, lost knee-jerks, Romberg's sign, Argyll-Robertson pupil, optic atrophy, etc.

The wife was 52 years old. She denied the existence of white blood in her family. She had had two children by a former husband, but had not been pregnant in her present marriage. About a year ago she began to suffer from sharp pains in the legs and trunk, and difficulty in walking. The diagnosis of tabes was based on these symptoms and on dimness of vision, Argyll-Robertson pupil girdle sensation, partial double ptosis, lost knee-jerks, Romberg's sign, etc.

Dr. H. J. Berkley, of Baltimore, said that he had not seen many cases of tabes in the negro and considered the disease an extremely rare one. On the contrary, the allied condition of dementia paralytica in the negro is occurring in ever greater frequency, until at the present time, in Baltimore, the proportion of cases between negro and white paralytics is about the same in proportion to the general population. Before the war, dementia paralytica was unknown in the black race. It has only been since they have been subjected to what Krafft Ebing calls "syphilization and civilization" that the disease has appeared among them.

Dr. Charles W. Burr said that the man had been referred to in several papers on tabes in negroes read before this society. It is interesting that his wife now has the same disease.

Dr. J. Madison Taylor had known this man for 5 or 6 years. He considered it an error to regard these cases as pure negroes, as any one familiar with the negro would at once detect characteristic evidences of admixture in both cases.

Dr. F. Savary Pearce said that five years ago he had reported to this society the case of a white man who had had a sudden apoplectiform attack proving later to precede locomotor ataxia. This man's wife also had locomotor ataxia which had existed for thirty years previous to the husband's disease. Under these circumstances it seemed that syphilis could be ruled out of the etiology. While it is possible that a toxin might be transmitted from the man to the woman through insemination, one could hardly see how a toxin could be transmitted from the woman to the man in a case where there are no secondary lesions.

In a study of 194 cases which he had made in May, 1898, there were only two cases observed in the pure-black race, a proportion of about one per cent.; showing that the disease must still be regarded as rare among Africans. It is interesting to note that the colored man reported by Dr. Francine was one of the cases that for a time was much stimulated by testicular fluid injections (See Transactions Medical Society of Pennsylvania, 1896).

Dr. William G. Spiller said that the statement of Dr. Berkley that dementia paralytica is increasing among the colored people is of interest. It is the belief of many that dementia paralytica is almost invariably a syphilitic disease. In a recent discussion on this subject by the Pathological Society of London many of the speakers held this view. If both tabes and paralytic dementia are syphilitic diseases, the increase of dementia paralytica, and not of tabes, among the negroes is very peculiar.

TWO CASES OF PARKINSON'S DISEASE.

Dr. Wharton Sinkler showed two patients suffering from Parkinson's disease, in whom there were certain points of interest, and in whom the symptoms were rather atypical. The first patient was a man aged 53; a stable boss by occupation. He had never been unduly exposed to wet or dampness, and had sustained no violent shocks or injuries. About three years ago he began to notice a tremor in the right hand. This increased in constancy and intensity until the present time, and there is now a continuous and excessive tremor of the right hand. There is some tremor of the leg. The tremor involves the fingers, hand and forearm, but the position of the hand is not that which is characteristic of paralysis agitans. The tremor can be arrested by voluntary effort; for example, while grasping the hand of the examiner there is no tremor for several minutes, and the tremor can also be arrested by making some other movement of the hand; for example, patting the leg, as was the case in the patient exhibited by Dr. Spiller at the last meeting. There is no change in the facial expression, and there is no peculiarity of gait.

The other case was noteworthy because of the early age at which the disease began, and the intensity of the symptoms. The patient was a man of forty-five years, an architect by profession. Ten years ago, when thirty-five years of age, he slipped into an opening in the floor of a building under construction, and fell in as far as the hip of one leg. He had to be pulled out, but was apparently not injured at the time. He walked home without difficulty. Several months later he began to observe stiffness in the left leg, and some tremor in the hand. These symptoms continued, and the tremor extended gradually to all of the limbs. About three years ago it was accidentally discovered that there was a prominence in one of the lumbar vertebræ, and on showing this to the physician who was then attending him—a homeopath—an operation upon the spine was urged. A long incision was made, the scar of which remains, and the surgeon says that he removed a fractured transverse process of one of the lumbar vertebræ. The patient says that, although he was not benefited at all by the operation, since that time he has grown no worse. He presents every feature of a case of paralysis agitans. The expression is mask-like; there is constant tremor of both hands and legs; the flexed position of the hands is characteristic, and there is much rigidity of the body. In attempting to walk there is marked propulsion. The patient can run with great ease, stop suddenly and turn without trouble. He is unable, however, to stand. He can run up stairs two steps at a time with ease. Attempts to write are almost illegible, but if a ball is thrown to him, he catches it with great facility.

Dr. J. Madison Taylor said that he, in conjunction with a highly educated man interested in physical culture, had devised a series of exercises that had relieved some of the most serious features of Parkinson's disease. He did not claim to cure the disease, but to mitigate most satisfactorily the worst disabilities. So far as he knew, this had not yet been done satisfactorily, and he hoped to present these cases to the society for inspection.

THE PATHOLOGICAL FINDINGS IN A CASE OF GENERAL CUTANEOUS AND SENSORY ANESTHESIA WITHOUT PSYCHICAL IMPLICATION.

The detailed clinical history of this case, reported by Dr. Henry J. Berkley, can be found in *Brain*, Vol. XIV, Part IV, 1891. A careful search of the medical literature has disclosed no similar instance occurring either before or since the date of its publication.

A synopsis of the clinical record as then published shows: (1) A strong hereditary tendency to nervous instability, the mother, two maternal uncles, two brothers and two sisters having been insane. The father had died of tuberculosis. (2) A syphilitic infection acquired from her husband at the age of twenty-nine years subsequent to which the patient had several abortions, sore throat and falling out of the hair.

For twenty-two years after the subsidence of acute symptoms due to the syphilitic process there was fair health. Then came an attack of acute arthritis after which the patient never fully recovered her former physical condition. Within a few months thereafter her eyesight began to grow dim, there were sudden flashes of light before the eyes, and vision was gradually extinguished, only sufficient remaining to enable her to distinguish light from darkness.

In the early summer of 1889, nearly six years after the rheumatic attack, the patient began to experience a general tingling and formication in the skin of the entire body, which was shortly followed by several spells of uncontrollable vomiting.

A consensus of the numerous examinations made during the years 1889 and 1890 showed a total loss of thermic, pain, olfactory, gustatory, equilibrium, pressure and weight sensations; almost total loss of the visual sense; and a partial loss of tactile and muscular impressions, muscular sense and auditory perceptions. None of the special senses, or cutaneous sensations remained wholly uninvolved.

Besides these disturbances of the sensory apparatus several other symptoms of almost equal prominence were recorded.

The musculature, while responding to the will, did so in such a feeble manner that the patient was incapacitated from helping herself to any extent. Thus the dynamometer when taken in the hand and squeezed was so feebly compressed that the indicator showed no movement on the dial, though the woman exerted every effort in the trial. Despite this fact, however, both nerves and muscles responded promptly to the galvanic and faradic currents, nor was there anything abnormal noticeable about the quality or time of the reactions.

The cutaneous reflexes were all abolished. The faucial and pharyngeal reflex movements were absent, and a sound could be passed over the epiglottis into the larynx without eliciting a sensation of discomfort or inducing cough. During the attack of vomiting there was no sensation of nausea.

When first examined the knee-jerks were present and normal. The biceps-jerk could also be elicited with some little trouble. The reaction of the abdominal muscles was lost, and the ankle beat was feeble. By the middle of July, 1891, the right knee reflex had disappeared, and the left one was weak. All other reflexes, deep and cutaneous, had been completely abolished. A year later the jerk of the left patellar tendon was found to be extinguished.

Furthermore, with auditory perceptions a progressive dulling could be noted. When the woman was first admitted to the hospital the sense of hearing was fairly acute in both ears. Gradually the difficulty in receiving auditory impressions increased, until finally the voice could not be heard except with strenuous effort.

The ophthalmoscopic examination showed an extensive choroiditis pigmentosa with atrophy of the optic nerves. Only a few vessels could be seen in the retina, and these were of minute size. In both lenses there was a beginning cataract.

Whether the optic nerve atrophy should be considered part of the general disease-process, beginning as it did long before the other symptoms, is somewhat problematical, though in tabes the same trouble is not infrequently noted as a forerunner of definite symptoms of the disease.

The pupils were at first in a state of mid-dilatation, and responded slowly to light, direct or reflected. They did not dilate on irritation of the cervical sympathetic. At a later stage the pupils became somewhat narrower, but remained sluggish to stimuli.

There were a number of interesting disturbances of the glandular secretions. During the fall of 1889 the mouth was found to be almost absolutely dry, the tongue heavily cracked, the epithelium eroded from its tip and sides, while the entire buccal surface was red and congested. At the same time the secretions of the lachrymal glands had almost ceased, the surfaces of the conjunctivæ being dry and injected. The skin was also dry and devoid of odor. After a course of potassium iodide these phenomena slowly abated, and the secretions returned to a more normal state.

Various unusual sensations troubled the patient at times. Cramps of the gastrocnemii, sensations of burning between the scapulæ, a girdle feeling around the abdomen and neuralgic twinges in the nerves of the extremities were often complained of. Occasionally there were clonic fibrillary spasms of the small muscles of the thumbs.

Attacks of diarrhea, refractory to treatment with drugs, though yielding to a continued milk diet, occurred in the latter part of the winter of 1890. These were on several occasions followed by a protracted vomiting without pain or nausea.

Trophic symptoms in the form of a bulbous appearance of the finger tips and ridging of the nails, together with purpuric spots developed late in 1891.

Throughout the long course of the illness there was never the slightest departure from normal mentality on the part of the patient; no hysterical attacks, no pathological depression or exaltation, and no delusions or hallucinations were ever noted. A slight apathy was the only perceptible change in the mental phenomena, but this was not greater than is frequently noticed in those who have become blind and helpless.

The patient's condition did not materially alter after the record of the case was published. The anesthetic and other symptoms, from the reports of the house physicians to the hospital, remained about stationary, and on the rare occasions on which Dr. Berkley saw her there were no additional phenomena to be noted other than a steady though slow decline of the vital powers. Late in the year 1893, another ophthalmoscopic examination was made by Dr. H. Friedenwald, who found in the left eye an extensive and typical retinitis pigmentosa, the papilla being blurred and of a dirty yellow color. Few vessels could be seen. In the right eye the clouding of the lens was so profound that the retina could not be seen.

About the middle of May, 1898, Mrs. R. became slightly lethargic, a condition that slowly increased to coma, in which state she died on the 25th of the month.

The autopsy, performed ten hours after death, was distinctly negative, all portions of the central and peripheral nervous systems showing an apparently natural condition. The larger vessels of the thorax, abdominal and cranial cavities, showed scattered atheromatous plaques, but were not considerably thickened. The right middle cerebeller and both posterior communicating arteries of the circle of Willis were congenitally rudimentary. There was some gelatinous thickening of the pia over the central regions of the hemispheres. The optic nerves showed but faint signs of a diseased condition, although the left nerve was a little smaller than the right one. Both kidneys were atrophic, weighing 100 grams each, the loss being principally in the cortex.

After proper hardening for the various Nissl, Weigert, Marchi and other stains, sections of the entire nervous system were made and studied. The results obtained were to a degree remarkable, and for the sake of convenience may be separated into three categories: (1) Lesions appertaining to the blood vessels; (2) those of the proper nerve elements, and (3) those belonging to the membranes surrounding the encephalon and cord.

The fundamental pathological basis for the various nervous phenomena described in the clinical history of the case is as follows: (1) An hyaline-fibrous degeneration of the arterial system existed, which was not confined to the central nervous regions, but was equally evident in the roots of the spinal nerves, and in the skin tissues. The degree of alteration varied from slight thickening of the muscular layer to complete closure of the lumen from hypertrophy of the middle coat of the vessel walls. The morbid change was accordingly not uniform, but reached its maximum of intensity in the vessels of the lower dorsal cord, the meninges of the bulb and cord, and also in certain of the root bundles of the cerebro-spinal nerves. (2) As a consequence of the vascular lesions there were degenerations of an atrophic order in the nerve cells of the gray horns of the cord, more particularly in those of Clarke's column, and in the medulla oblongata. To this alteration in the central nervous substance at least a portion of the various symptoms must be attributed.

Besides the principal lesions there were others of considerable though minor importance.

As an explanation of the numerous symptoms of the case it would appear most reasonable to suppose the existence of a disease process affecting simultaneously both the peripheral and central nervous systems. Assuming that the arterial lesions were of late specific origin—and of this there can be but little reasonable doubt—a chronic progressive involvement of the nutrient channels, here and there leading to marked narrowing, or even to closure of isolated vessels, might certainly have produced just such a train of symptoms as was present. Of primary importance would be the involvement of the arteries of the anterior and posterior nerve roots of the bulbospinal system, though the direct lesions of the nutrient supply to the terminal nerve apparatus, which have to be inferred as they could not be definitely determined, would be of equal value. This inference of the implication of the end-apparatus is justifiable since the arterial degeneration in the skin was quite extensive, and whenever there is starvation of a nerve tissue there result pathological reactions which may be manifest in a multitude of ways.

The slowly progressive character of the symptoms is also consistent with the theory of tissue starvation. All the lesions of the nerve cells of the cord and bulb are of this type—atrophy and pigmentary degeneration from malnutrition. Whenever nutrition is at a low ebb, metaplastic granules accumulate in the protoplasm of the cell.

The condition of the optic tracts—in which the lesions are identical with those of other nerve roots except that they are more advanced—would argue that the thickening of the blood vessels was of long standing, and that only when the process had advanced to an extreme degree did any definite symptoms show themselves. This is exemplified more particularly in the state of the cortex cerebri. Though in this region vascular disease was manifest and diffuse, it had not advanced nearly to such a degree as in the gray matter of the dorsal cord or in the adjacent meninges. As a consequence, the functions of the cortex, while not as perfect as in youth, were not reduced to the same low level as those of the cord and bulb.

One pathological fact should be remembered in considering the clinical symptoms, namely, that it is not necessary for a vascular lesion to proceed to such a profound degree as to cause the entire shutting off of the nutrient supply before a nerve tissue will show signs of deviation from its normal functions. With a reduced supply of nutrient plasma, definite manifestations of nervous exhaustion are brought about, and these are not due to a degeneration of the component portions of the neuron, which is visible in the tissue after death by our present methods of preparation, in the form of morbid alterations of the cytoplasm, axone, or myeline. Long before this stage is reached the entire neuron is incapable of performing its natural functions in an efficient manner, and as a consequence, anesthesias, paresthesias, diminution or exaltation of the reflexes, and dulling of the special senses can be noted. Almost precisely similar results are encountered in advanced stages of progressive paralysis, especially in the syphilitic cases, in which, when vascular lesions of the arteries of the cerebrum and cord have advanced to a profound degree, there is a gradual but progressive dulling of cutaneous sensibilities and special sensations.

Dr. C. W. Burr exhibited a case of general anesthesia.

Dr. F. X. Dercum remarked that while the interpretation which Dr. Berkley had given was exceedingly interesting, it did not seem entirely satisfactory. In cases where there is great vascular degeneration, as in pronounced arterial sclerosis, the sensory symptoms found in Dr. Berkley's case do not occur, and therefore the symptoms could hardly be ascribed to the paucity of the blood supply.

It seemed to him that this case would suggest the presence of a toxin. The eccentric position of the nucleus and nucleoli, among other things, would suggest this, and the changes in the blood vessels and kidneys would be in keeping with such a view.

Dr. Joseph Sailer said in reference to the statement that double nucleoli in a nerve cell were indicative of an irritative lesion, that he had repeatedly found these double nucleoli, and in looking up the subject, found that they had been noted by others in cells presumably normal.

Dr. W. G. Spiller said that a few cases of general anesthesia, and some with necropsy, had been reported. He had observed a case of this character in the service of Dr. Dejerine at the Salpêtrière, and had been permitted by Dr. Dejerine to study the specimens from another case reported later by Dr. André Thomas. In the latter case no such arterial disease as in Dr. Berkley's case was observed. There was, however, an extraordinary condition of the central nervous system: the cerebellum and spinal cord were small, and the posterior columns markedly degenerated. Dr. Thomas ascribed the general anesthesia to hysteria.

Dr. Spiller thought that if the general anesthesia in Dr. Berkley's case were produced by the vascular lesions, we should expect to see general anesthesia in other cases of vascular disorder, as in syphilis. In some such cases there is extreme degeneration of the blood vessels with great impairment in the nutrition of the tissues, yet we do not find persistent and general anesthesia in such cases. Dr. Berkley's report was a most important and interesting one.

Dr. Henry J. Berkley said that while a number of other cases of general cutaneous and sensory anesthesia had been reported, there were none in which the pathological examinations made disclosed the same anatomical findings as in his case. His case stood in this respect isolated in medical literature.

With reference to double nucleoli, he said that these were found normally in some of the lower animals. He doubted the correctness of the statement that they are found normally in the human being. In many thousand sections he had never found them except where there were evidences of an irritative process.

Dr. Berkley could not agree with the suggestion of Dr. Dercum as to the condition being due to a toxic cause. While the atrophy of the kidney and of other tissues might induce the presence of more or less toxic material, yet in a case where there is such immense alteration of the blood vessels, and these occurring much better developed in one locality than in another, he did not think it necessary to seek any other cause for an explanation of the symptoms. In any tissue that is half starved, one always gets nervous symptoms. Lesions of the media are more liable to produce a symptom-complex corresponding to the phenomena in the present case than when there is alteration in the other coats. When the hypertrophy of the middle layer is very great it almost completely stops the circulation of the nutrient plasma.

BRAIN FROM A CASE OF CONGENITAL ABSENCE OF THE VISUAL TRACTS.

Dr. W. G. Spiller exhibited the brain from an idiot, supposed to be about twenty-two years old. The palpebral fissures of the patient were very small and no indications of an eyeball was seen in either orbit. Not the slightest opening in the cranium or the passage of an optic nerve could be found. The optic nerves, chiasm and optic tracts were entirely absent. No external geniculate body could be seen on either side. The pulvinar was considerably smaller than in a normal brain, and no indications of an optic tract passing from it was found. The anterior colliculi of the corpora quadrigemina were of normal size, which would seem to indicate that these structures in man are not an essential part of the visual system. The cuneus on each side was very small, although a calcarine fissure was present.

60. UEBER EINEN FALL VON KATALEPTIFORMER LETHARGIE MIT SIMULATION VON CHYLURIE (Cataleptic Lethargy, with Simulation of Chyluria). M. Rothmann and A. F. Nathanson (Arch. f. Psych. und Nervenkr., XXXII., 1., p. 283).

A very interesting case of hysteria in a girl of 19 is here reported. The patient brought about a condition of seeming chyluria by practicing intravesical injections of milk. Besides this, she was attacked by cataleptic seizures, accompanied with lessening of the area of liver dulness, diminution of urea and presence of ammoniacal urine. Glancing over the clinical history and experimental observations made on the case, the authors came to the conclusion that the attacks were due to disturbances of metabolism in the liver, and especially of the urea-producing functions. JELLIFFE.